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THE DIAGNOSTIC SIGNIFICANCE OF THE MITRAL PRESYSTOLIC MURMUR.[1]

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THE characteristics of the typical mitral presystolic murmur are well known and ought to be easily recognized. It is a loud, long sound, rough and vibrating in quality, commonly with a well-marked thrill. It is audible over a fixed and limited position—the mitral area—a circle of about an inch around the point where the apex touches the thoracic wall; the direction of the sound being toward the apex. Its maximal intensity is to the right of the apex beyond where the blood current enters the ventricle. It may be heard indistinctly above the third rib and, very exceptionally, in other directions. Its rhythm is at the end of the long pause immediately preceding and abruptly ending at, and with, the first sound. The rhythm of the presystolic murmur, when the pulsations are slow, is easily recognized by feeling the carotid pulse while auscultating, because the pause before the first sound is much longer than that which follows it. But when the beats are more

[1] Read before the Association of American Physicians, September 18, 1888.

rapid the distinction cannot be drawn, for the increased pace is gained at the expense of the period of rest, and one pause may be as short as the other (Fagge). The designation presystolic is sufficiently appropriate, since the murmur immediately precedes the ventricular systole. These important facts show that it is found during the diastole of the ventricle. The closure of the sigmoid valves, the filling of the ventricles, and the contraction of the auricle, take place during this period of the heart's revolution. With the murmur we often hear pronounced accentuation of the first sound at the apex, and of the second sound over the pulmonary artery, and, also, when it has existed for some time, a reduplication of the second sound.

The blood in the lesser circulation rushes on through the distribution of the pulmonary arteries into the elastic pulmonary veins, thence directly onward through the auricle into the ventricle, continuing its flow until the ventricle, as well as the auricle, is nearly full, when suddenly the auricle contracts on its contents and completes the distention of the ventricle previous to its systole.

Ludwig and Hess showed that the mechanism of the closure of the left auricular-ventricular orifice does not reside in the valve alone; the surrounding muscles of the ventricle have an active share not merely in floating up the valve curtains, but in reducing the size of the aperture which these valves have to close. The papillary muscles keep the curtains tight during the contraction of the ventricle. The whole action of the heart is screw-like, six or seven distinct layers of muscular fibres crossing one

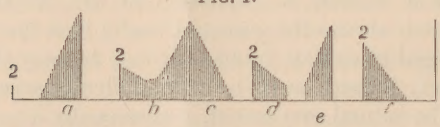
another. The base muscles do their share of the work of closure, the valves promptly complete it. When the muscles of the base are enfeebled, the valve curtains are insufficient to close the orifice (MacAlister).

It is a mistake to suppose that the presystolic murmur is always the same. Usually it is the most prolonged of cardiac bruits. It may be very short; so short, indeed, as to be with difficulty separable from the natural first sound. Occasionally it appears like a tone or accentuation of the first sound. In this case the first sound becomes so sharp and clear that it may easily be mistaken for the second sound, which becomes inaudible at the heart's apex, and, unless careful attention be paid to the rhythm, there is danger that the murmur may be mistaken for the normal sound, instead of an abbreviated presystolic bruit.

It is a very variable sound both as to its length, its loudness, and its quality. It is heard at some times and not at others. It may, and indeed often does, follow immediately the second sound, and continue up to the culmination of the first sound, with which it suddenly ceases. It is generally more intense at the ending than at the beginning. Traube says that, if the action of the heart be retarded by digitalis, this murmur ends before the first sound. Flint states that only in one of his cases did the murmur cease before reaching the first sound. It often commences in the middle of the diastole with the auricular systole, and runs up to the first sound. Again, it lessens in volume at the middle, and, rising, ascends to its first intensity.

It sometimes becomes altogether inaudible in the middle of the long pause, and then reappears. In some cases there is an entire absence of a murmur, but instead we hear an accentuated clicking first sound.

FIG. 1.

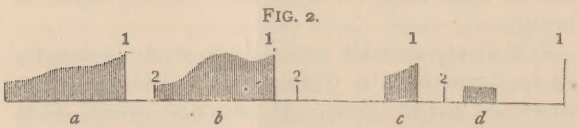


Typical and other varieties of presystolic and post-diastolic murmurs. 1. First sound. 2. Second sound.

a. Presystolic murmur (typical).
b. Presystolic murmur united with post-diastolic.
c. Systolic (mitral insufficiency).
d. Post-diastolic, separated from presystolic.
e. Presystolic. *f*. Post-diastolic.

(After Sansom.)

FIG. 2.



1. First sound. 2. Second sound.

a and *b*. Continuous post-diastolic and presystolic murmurs.
c and *d*. Irregular presystolic.

(After Gairdner.)

The name of post-diastolic is given, very inappropriately, to the murmur heard immediately following the closure of the sigmoid valves. This sound, though rough, is not blubbering and vibrating. It is generally indicative of pronounced stenosis. The diastole of the ventricle begins with the closure of the sigmoid valves. A bruit that immediately follows the second sound, which, it is ad-

mitted, is found at this precise period of the cardiac rhythm, ought to be designated diastolic without prefix of "post." These two murmurs, the presystolic and post-diastolic, may be present independently of one another, and when found in the same patient may coalesce, or they may be separated by an appreciable interval.

Although much has been written of late about the presystolic murmur, yet all who have had much experience in consultations in cardiac cases will confirm the statement of Broadbent, that it is not infrequently, even now, entirely overlooked and often mistaken for the mitral systolic murmur, the differential diagnosis of which has been clearly established. Formerly it was supposed that the presystolic murmur was rarely found, but closer observation has shown that it is of frequent occurrence, especially among women and children. Flint observed it in sixteen out of thirty-nine cases of mitral disease. Its grave prognostic significance alone justifies the attention that has been given to it.

In this paper, I shall only detain the Association by a brief study of the factors entering into the production of this interesting murmur when of organic origin.

There must be two factors in the production of "fluid veins," whether they are obstructive or regurgitant: the lesion, and the propelling force, together with the other well-known influences, modifying blood pressure. For the generation of sonorous fluid veins, we must have the passage of a jet of blood forced through a narrow constriction into a wider cavity or part of a vessel.

It is acknowledged that mitral stenosis is the lesion in a very large majority of cases in which a presystolic murmur is heard. Many prominent authorities believe that this lesion is invariable. The constriction is frequently found at post-mortems in cases in which there had been no presystolic murmur detected during life.

The mitral valves and the auricular-ventricular orifice are specially liable, in early life, to valvulitis from acute or subacute rheumatism and other causes, such as scarlet fever, measles, or chorea. These leave formations of connective tissue which narrow the orifice itself in the annular forms or by diaphragmatic partitions with buttonhole orifices, or the leaflets adhere together and produce funnel-shaped constrictions. In some instances the valves lose completely their natural formation. These new conditions are most favorable to the production of murmurs, which are due to oscillations in the blood itself. The current of blood passes through a small opening into a wider cavity, and is divided up into a number of jets, forming fluid veins. The rapidity and force of the current must cause variations in the intensity of the sounds.

The obstructive lesion of the mitral orifice, it must be borne in mind, is of very gradual formation, of months or of years. It commences often insidiously. The auricular-ventricular orifice is so large that it can bear some lessening of its size without seriously interrupting the proper filling of the left ventricle. Thus stenosis may be sufficient to produce the vibratory sonorous murmur, without any marked subjective symptoms.

While admitting that stenosis of the mitral orifice is, in a very large proportion of cases, the pathological lesion met with where this murmur is found, yet there are numerous cases reported in which the mitral orifices were not altered in size, but in which other obstructive and narrowing conditions were found inside the ventricle—such as shortened chordæ tendineæ, thickenings and rugosities about the leaflets of the valves or in the walls of the ventricle, to account for the production of the characteristic prolonged, blubbering, presystolic murmur. Wherever and whenever we find physical conditions to produce "fluid veins" with vibratory phenomena, we can have the same sound. We need not recall Flint's three cases in which aortic insufficiency, as demonstrated at post-mortems, produced functional murmurs with the characteristics of the mitral presystolic murmur. Dr. Bramwell has published such a case, and Dr. Guitéras reported three. Sansom speaks also of cases somewhat similar to Flint's. Osler has had a well-marked instance.

The writer has never clinically met with such a case, but he has heard aortic diastolic murmurs where there were marked accentuation and shortening of the first sound, similar to the effects met with in cases of mitral stenosis. While it is true that such cases have been reported by the most competent auscultators, they are in fact but rarely met with. There is, however, no *a priori* reason why they could not exist. Aortic regurgitation murmurs are diastolic in time, and if, as Flint claims, the leaflets of the mitral divide the reflow current in the same manner as in the production of the murmur at the auricular-

ventricular orifice, the sounds may be formed and a prolonged murmur of presystolic rhythm with the blubbering vibrations may be heard. Dr. Guitéras maintains that this functional presystolic mitral murmur is more frequently heard than Dr. Flint supposed. Balfour, Nixon and Sansom, and others think differently. There can be no doubt that the diastolic aortic murmur can be conducted to the apex by the solid walls of the hypertrophied ventricle, the current of blood itself as well as by the sternum and walls of the chest. The infrequency of these functional mitral murmurs, when there is an aortic insufficiency, is difficult to explain.

Dr. Balthazar Foster suggests an explanation of how this murmur is heard best at the apex: That different valves are affected in different cases, and that, if the left sigmoid near the mitral is affected, the blood falls directly back into the ventricle and the bruit is heard best at apex; whereas, if the right or posterior sigmoid valve is diseased, the regurgitant blood impinges on the septima and the bruit is carried down the right side of the heart. Dr. Guitéras maintains that these propagated murmurs are in fact mitral obstructive murmurs, and that they are more apt to develop when the posterior aortic segment is affected, because in such cases the recurrent stream is brought directly against the anterior leaflet of the mitral valve.

Dr. Bramwell (*Diseases of the Heart*) refers to a case of rupture of the posterior coronary segment of the aortic valves in which no presystolic murmur was audible. There must be some unusual physical condition which has not yet been appreciated.

There is also a murmur which Barlow, Leaming, Flint, and Donaldson have designated as intraventricular, which, when there is much hypertrophy of the left ventricle, seems to occupy the first portion of the systole. This resembles the qualities of the presystolic, and, when the heart is beating rapidly, may be confounded with it. Of course, in diagnosticating mitral stenosis, everything is taken into consideration, together with the murmur.

Let us inquire from what sources the necessary force of the blood-current is derived. Dr. Gairdner taught that it is produced by the systole of the left auricle, and he accordingly named the adventitious sound the "auriculo-ventricular murmur." This could not possibly apply to the sound which exists before the contraction of the auricle, which, in health, occupies one-fifth of a second of time, and immediately precedes the systole of the ventricle. The feeble force of the auricle in health was shown by Chauveau, who, by prolonged irritation, exhausted its contractility, and found that the ventricle continued to act effectively, and kept up the circulation. Ludwig confirmed this result by introducing a tube through the auricle. In fact, the auricle may be regarded as the dilated termination of the pulmonary veins, acting as a reservoir to supply the ventricle, and to regulate the blood-pressure in it. The auricular cavities are never completely obliterated during contraction, though this does not appear to be the case with the auricular appendages.

We may ascribe the production of the presystolic murmur, in beginning obstruction of the mitral orifice, to the force of the left auricle. Temporary

compensation is promptly established in the force of the circulation. It seems that there is a reserved force, as in involuntary muscles, which is called forth in the emergency.

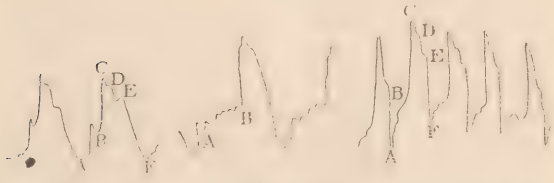
We further admit that, as the result of obstruction at the mitral orifice, impeding the influx of blood into the left ventricle during the diastole, the left auricle, having extra work to perform, does become first dilated and subsequently hypertrophied. Indeed, percussion, in rare instances, reveals abnormal bulging over the auricle, and frequently the pulsation of the auricle is perceptible about the fourth rib. The walls of the left auricle, which are normally about one and a half lines in thickness, may be increased to as much as a quarter of an inch, with proportionate augmentation in strength. It may be that the prolongation of this bruit is due to this hypertrophy, but it is almost incredible when we know that it is the longest, as well as the most irregular and vibrating, of all cardiac murmurs. We question whether it is a force sufficient by itself to overcome the resistance caused by advanced mitral constriction, and to give us the acoustic characteristics of the presystolic murmur.

It must be borne in mind that the auricular systole does not occur until the very last portion of the diastole, and the murmur often begins after the second sound, and includes the so-called "post-diastolic." These phenomena occur previous to the auricular systole. We readily admit that its influence is shown in reinforcing the intensity of these murmurs.

In mitral stenosis the auricular portion of the

cardiagrams is of longer duration, and its tracing higher, because the left auricle is dilated, and contracts with force, emptying itself with difficulty; it often shows a series of wavelets, giving it a serrated appearance corresponding to the purring thrill felt by the hand. Ott, Hess, and Galabin attribute the late closure of the pulmonary valves to the diminished elasticity of the artery in consequence of long-continued high pressure.

FIG. 3.



a Mitral stenosis. Pulse rate 80 (after Galabin).

b Mitral stenosis. Long, rough, presystolic murmur. Pulse rate 57 (after Galabin).

c Normal curve from man (after Landois).

A B. Auricular systole.
B C. Ventricular systole.
D. Aortic closure.
E. Pulmonary closure.
E F. Ventricular diastole.

The cardiagram of the normal heart (see Landois's *Physiology*) proves that the aortic valves close a little before the pulmonic valves. They close, however, so near together that only a single sound is heard. In mitral stenosis (stage of non-compensation) the aortic valve closes sooner, and the pulmonic valves later than normal. This is because the left ventricle contains less blood, and the right more than nor-

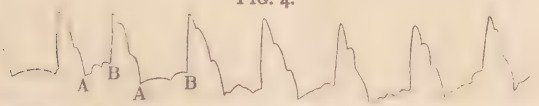
mally. There results what is called a reduplicated second sound, which is an increase of a physiological condition. All conditions which cause the aortic valves to close rapidly (diminished amount of blood in the left ventricle), and the pulmonary valves to close later (congestion of the right ventricle)—both of which exist in mitral stenosis, favor the production of a reduplicated second sound, which occurs in one-third of the cases of mitral stenosis.

If mitral contraction occurs at a very early period of life, whether as a primary affection, or secondary to a corresponding narrowing of the tricuspid, other parts of the circulatory system become accommodated to it, and the patient may reach adult life before the lesion produces any symptoms—the post-mortem revealing the fact of the organic defect. Dr. Wilkes reports cases in which resulting hypertrophy of the right side in children was so great as to cause bulging of the chest, and in which the pulmonary artery partook of the hypertrophy, and became thicker than the aorta. On the other hand, cases occur in which, owing to mitral stenosis early in life, so small a quantity of blood is thrown into the left heart that there is imperfect nutrition of the whole body, which accommodates itself to the defect. Hypertrophy of the right ventricle and the left auricle compensates so thoroughly for the deficiency, that there may be no subjective symptoms, and no bruit, perhaps, beyond an imperfect first sound.

Dr. Gairdner did not ascribe the production of the presystolic murmur wholly to the muscular contraction of the auricle. Dr. Wilkes held that the

murmur might anticipate the auricular systole, and that it might occur not only at that time, but also during the heart's diastole and pause. Dr. Galabin came to a like conclusion from the evidence afforded by the cardiograph. Dr. Sansom claims that the causation of the presystolic murmur may be independent of the auricle; he states "that in many cases he had observed that though there had been a prolonged presystolic murmur commencing in the long pause almost immediately after the second sound, cardiographic evidence has shown the auricular systole to occupy its normal position just anterior to the commencing contraction of the ventricle." He cites a case in which a murmur occupied at one time a portion and at another almost the whole, of the long pause, and the autopsy showed that the auricular systole could have had no share in producing such a murmur, for not only was the left auricle so dilated

FIG. 4.



Sansom's case, showing mitral stenosis, while auricular systole is in normal position. A B. Auricular systole.

that its walls could have exerted no appreciable muscular power, but it was lined by a closely adherent, old, laminated blood-clot. It thus appears that the post-diastolic murmur, as well as the presystolic, may be caused in the diastole, and be due to the entrance of the blood independently of the auricular systole, it being urged through the stenosed

aperture, owing to the tension under which it has been retained in the elastic and distended auricle and pulmonary veins.

We feel justified in concluding that the systole of the left auricle is a factor of greater or less force in the production of this mitral direct murmur, but it is not the only one, and the others may produce it independently of the auricular systole.

Negative pressure. Let us see whether any negative pressure is brought to bear upon the left auricle and pulmonary veins to assist in getting the current into the left ventricle. Is there any suction action of the left ventricle of the heart? Until the publication, in 1878, of Goltz and Gaule's experiments, the majority of physiologists attached little or no importance to any suction action of the heart, although in all times there have been zealous advocates of the doctrine that diastolic suction was an important factor in the circulation. Goltz and Gaule, by the use of ingeniously constructed maximum and minimum manometers, determined the maximum and minimum pressures in the left and in the right ventricle. They found with natural respiration, in the left ventricle of a dog, a negative pressure of 52 mm. of mercury. In the right ventricle there existed, in another case, a negative pressure of 17.2 mm. of mercury.

In accordance with the generally accepted ideas, they at first were inclined to attribute this negative pressure to the expansion of the chest during inspiration. To their great astonishment, however, they found, after the chest was opened, and independently of respiration, a negative pressure in the left

ventricle of 23.5 mm. of mercury. This latter negative pressure, therefore, cannot be due to the elastic traction of the lungs. They attribute it to the elastic resilience of the ventricle following the systole, like that after compression of an elastic balloon. They say: "The adherents of the doctrine, according to which the heart acts as a suction-pump, will find their boldest expectations surpassed when they hear that the suction force of the left heart of a large dog, at the beginning of the diastole, is equal to a column of water 320 mm. (23.5 mm. of mercury), and that this force in a healthy man is probably even much higher." (Goltz und Gaule: "Ueber die Druckverhaltnisse im Innern des Herzens," *Pflüger's Archiv*, 1878, Bd. 17, S. 100.)

In 1879, Moens published an article ("Die erste Wellengipfel in dem absteigenden Schenkelder Pulscurve," *Pflüger's Archiv*, Bd. 20, S. 517, 1879), in which he attempted to show that the negative pressure found by Goltz and Gaule existed, not during the diastole, but during the systole. In order to make clear how a negative pressure might be produced at this period of the heart's action, he takes an elastic tube open at one end and connected at the other by means of a stopcock with a pressure-bottle. If the stopcock be suddenly closed while the fluid is flowing from the bottle through the tube, there is a negative pressure produced in the tube, beginning near the stopcock. Likewise, in the left ventricle, he argues that, as the blood is shot out by the ventricular contraction into the aorta, a negative pressure is produced in the ventricle. His chief

inference, that the negative pressure is present during the systole and not during the diastole, was because a minimum manometer, passed down the jugular vein into the superior vena cava, failed to register negative pressure, as would be the case if the right ventricle exerted diastolic suction. He regards this experiment as proving that the negative pressure is not during the diastole, otherwise there would be a suction force exerted on the blood in the right auricle and vena cava superior. If the negative pressure is not during the diastole, it must be during the systole.

It will be observed that Moens does not actually demonstrate that the negative pressure is during systole. He infers it indirectly. On the other hand, he seems to have overlooked the fact that Goltz and Gaule found a negative pressure in the right auricle of 11.2 mm. mercury (this might have been due to inspiration), the negative pressure in the right ventricle, in the same case, being 17.2 mm. mercury. Moens is quoted by Martin and Donaldson, Jr., as having shown that the negative pressure exerts no suction force on the blood in the auricles.

In consequence of the doubt which Moens's work raised as to the period at which negative pressure existed in the ventricles, de Jager, in 1883 ("Ueber die Saugkraft des Herzens," *Pflüger's Archiv*, Bd. 30, S. 491), published experiments on this question, and came to conclusions in opposition to Moens's and in confirmation of Goltz and Gaule's. De Jager determined that the pressure at the beginning of the aorta never becomes negative, as it should be in case the negative pressure in the ventricle were due to

the suction of the column of blood poured out from the ventricle. He also found that with the chest open, so that the action of inspiration on the aspiration of blood into the right heart was removed, the pressure in the right auricle becomes negative. He found negative pressures in the right auricle of 2 mm. to 6 mm. mercury. He does not believe that the auricles themselves are capable of exerting any suction force, and he, therefore, concludes that this negative pressure in the right auricle can be interpreted only as evidence of a suction exerted by it, which, of course, can occur only during diastole. The same conditions would, of course, obtain for the left cavities of the heart, as the experiments were made with the chest open. De Jager, therefore, concludes that a strong diastolic suction force is exerted by the ventricles.

As regards the causes of this suction force he mentions several possibilities, without coming to a decision. These possibilities are:

1. The elastic force developed during the systole, as in the compression of a rubber balloon.

2. The suction force on the exterior of the heart resulting from the negative pressure in the thorax (or the elastic traction of the lungs). This, of course, can be only one element, as negative intraventricular pressure is found after the thorax is opened.

3. Shortening of the large arteries coming from the heart.

Brucke inclines, as do Goltz and Gaule, to consider these factors as the essential ones.

Gruenhagen holds that Goltz and Gaule and de

Jager have established the fact of a diastolic suction action of the heart.

The experiments of Martin and Donaldson, Jr., however, on the mammalian heart, at the Johns Hopkins University, are opposed to the existence of any suction action of the heart. Their experiments are exact, and, it would seem, quite conclusive against the suction theory. It is difficult, however, at present, to reconcile them with Goltz's, Gaule's, and de Jager's experiments. They have the merit of putting the matter to a direct test, whereas the other experiments do not actually demonstrate a suction action, but leave it as an inference. They say:

"The negative pressure proved by Goltz and Gaule to occur in the ventricles for a brief period at the end of the systole, had already been shown by Moens not to affect the auricles, and, therefore, to be without effect in making the heart a suction pump as far as the venous system was concerned.

"Once the 'aspiration of the thorax' has been eliminated, the right auricle of the mammalian heart will not receive blood unless supplied to it under a decided, if small, positive pressure. While the heart in the closed thoracic cavity may, and probably does, act as a suction pump, this is not due directly to an active expanding force of the heart, but is the secondary result of the pneumatic conditions prevailing within the normal closed chest cavity."

If a suction action exists, it is evidently a factor not to be neglected in the study of mitral stenosis. All that increases the suction action, increases the

force with which the blood passes through the narrowed mitral orifice and doubtless increases the murmur. De Jager found that when the action of the heart became feeble—as after long exposure, or by cooling, or by loss of blood—then only a slight negative pressure, or none, was produced in the ventricles. We may, therefore, infer that all circumstances which enfeeble the action of the left ventricle diminish correspondingly the force of suction, and, therefore, the murmur. Such circumstances are failure of the heart's force from any cause, among which may be noted atrophy of the left ventricle in pure mitral stenosis. In general, whether the left ventricle is atrophied or not, the small amount of blood which it receives through the narrowed mitral orifice during non-compensation must be attended with a weaker systole, and, consequently, diminished suction and lessened murmur. All this line of argumentation, of course, is not pertinent, if we accept the doctrine that no suction force is exerted by the ventricles.

Let us further inquire whether any other force is connected with the passage of the blood through the left ventricle. We hear of the blood's passive flow through the mitral orifice. Has it no power? The right ventricle, which propels its contents with a strong wave through the pulmonary arteries and with comparative ease, owing to the absence of tonus or resistance, and of vasomotor influence to contend against, forces the blood into the pulmonary veins and through the left auricle into the ventricle. If it meets with any obstruction, the backward pressure would, of itself, add to the onward blood pressure in

the pulmonary veins, although the right ventricle promptly exerts its reserved compensatory force. If the obstruction continues, the extra work of the right ventricle gradually produces permanent hypertrophy which keeps up the compensation. When this is broken, there results the disastrous backward flow of venous blood with all its unpleasant consequences upon the lungs, liver, and other internal organs. This occurs, notwithstanding the protection afforded to the process of intra-pulmonary respiration, as shown in the safety-valve construction of the tricuspid valve (King), of the pulmonary semilunar valves (Flint, Jr.), and of the obstruction safety-valve principle of the pulmonary valve (Guitéras). The increased tension at the pulmonary valves gives accentuation of the second sound. Nature further provides a relief to the lungs by the increased capacity of the right cavities by from one-tenth to one-third over the corresponding cavities of the left side.

The right ventricle becomes very gradually hypertrophied. In some cases the enormously thickened walls completely cover the left ventricle and prevent our hearing the second sound at the apex and a very feeble impulse is noticed. The left ventricle, owing to the insufficient supply of blood through the auricular-ventricular orifice, is often not only not hypertrophied, but actually materially lessened in size—indeed, in a state of atrophy, unless there is also insufficiency of the mitral orifice as a complication. This additional lesion may cause hypertrophy of the left ventricle. May not the powerful right ventricle, by the blood pressure it causes in the pulmonary veins, be a prominent factor in causing variations in

the length and intensity of the post-diastolic and the presystolic murmurs in mitral stenosis?

If this hypertrophy be very marked, may it not be the propelling force producing the long sound commencing immediately after the systole and occupying the whole of the diastole? If the force be feeble, it may only produce the post-diastolic and cause the deficiency in the middle of the distance between the second and first sound. When the patient is in the recumbent position and the heart is quiet the murmur is often inaudible; on changing the position it may be heard. Active exercise increases in marked degree the intensity of the murmur. When dilatation succeeds, hypertrophy of right ventricle and tricuspid insufficiency take place—the forces are not powerful enough to cause the murmur. The auricular systole, if dilatation has not made the walls too feeble, may give rise to the presystolic. The increased power of the hypertrophied left auricle and right ventricle certainly would cause the fluid veins to be audible vibrations in obstructive mitral disease. As the muscular power leaves the excessively enfeebled patient, the sound utterly disappears. Indeed, it is very remarkable how the murmur sometimes disappears, temporarily or permanently, the condition of valve being unaltered.

To sum up (says Dr. Galabin, in his elaborate and able paper on, "Interpretation of Cardiographic Tracings," *Guy's Hosp. Reports*) the inferences suggested by a general review of all the tracings, it appears that the evidence of the cardiograph is in favor of the view that two totally distinct murmurs may be caused by mitral contractions: first, the

auriculo-systolic bruit, which may either run up to the first sound or be separated from it by a short interval; and, secondly, a diastolic bruit, due to the venous flow through the narrow and roughened orifice, which in rare cases may be blowing in quality and separated from the succeeding systole by a long pause; and that, thirdly, these two may be merged together into a compound murmur somewhat rough from its commencement, but much intensified in loudness and harshness toward its conclusion.

Marey, in some experiments by means of which he reproduced the various sounds and murmurs of the heart, the auricle of which was not contractile, introduced a plug perforated by a hole between the auricle and ventricle, and then found that a diastolic murmur was produced, but only when the auricular pressure exceeded a certain point. Hence, he concludes that the murmur of mitral stenosis may be either diastolic or auriculo-systolic according to circumstances.

How far is our estimate of the pressure in the pulmonary veins justified by experimental research? From this source we possess but little positive knowledge. Beutner seems to be the only authority who has published any actual measurement of the blood pressure in the pulmonary veins. He found a positive pressure of 10 mm. of mercury in the pulmonary vein of a cat. In the pulmonary artery of a cat he found a pressure of 17.6 mm. mercury. These pressures were, of course, measured with the thorax open, so that we cannot attach absolute value to them. They indicate however, that, relatively to the pressure in the pulmonary artery, the pressure in the pulmonary

vein is high, the pressure in the veins being more than one-half that in the artery. The pressure in the pulmonary vein, according to Beutner's measurement, is, moreover, absolutely a high one when compared with the very low pressure in the veins emptying into the right auricle. The pressure which Beutner found in the pulmonary artery is much lower than has been found by subsequent observers. The accepted belief is that the pressure in the pulmonary artery is one-third of that in the aorta; Goltz and Gaule regard it as two-fifths of the aortic pressure. If the ratio which Beutner found between the pressure in the pulmonary vein and that in the pulmonary artery holds true, then we may conclude that the pressure in the pulmonary vein is even higher than his observations indicate.

Inasmuch as actual measurement of the pressure in the pulmonary vein can be made only when the thorax on one side is open, the question arises: What influence upon this pressure has the elastic resilience of the lungs under the normal conditions of respiration? If the walls of the pulmonary artery and those of the pulmonary veins were of the same thickness, then, inasmuch as both vessels were subjected to the same intra-thoracic pressure, it is evident that changes in the intra-thoracic pressure, corresponding to the different phases of respiration, would not influence the blood pressure. The walls of the pulmonary veins, however, are thinner than those of the pulmonary artery, therefore the aspiration of the thorax must tend to lower the pressure in the pulmonary veins more than in the pulmonary arteries. It is impossible to say how great is this

influence of the elasticity of the lungs upon the pressure in the pulmonary veins.

The feeble tonus of the vessels in the pulmonary circulation is of the utmost importance, as changes in pressure in the pulmonary veins are quickly manifested in the pulmonary arteries and *vice versa*. This circumstance justifies us in assuming that change of pressure in the pulmonary artery is attended by corresponding changes of pressure in the pulmonary veins, so that changes in the force of contraction of the right heart exert a much more direct influence upon the circulation of blood in the pulmonary veins than in the left heart and the systemic veins.

Cohnheim says: "The blood in the pulmonary veins is, under physiological conditions, not like that of the systemic veins under negative pressure when it enters the heart, but is under a positive pressure which is only a little less than that of the blood in the pulmonary artery." (*Vorlesungen, über allgemeine Pathologie*, Bd. 1, S. 28, Berlin, 1882.) This statement of Cohnheim does not seem to rest upon actual measurement, but to be based upon the slight resistance offered to the blood in the passage from the pulmonary artery to the pulmonary vein, and upon Beutner's measurement with open thorax. He found the pressure in the pulmonary vein more than half that in the pulmonary artery. As no means have been found which enable us to measure the pressure in the pulmonary veins under natural conditions, we are left to such inferences as the above.